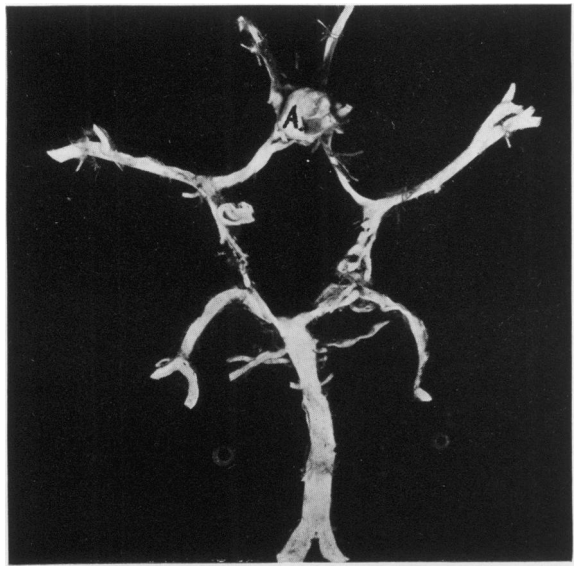

Fig. 1.

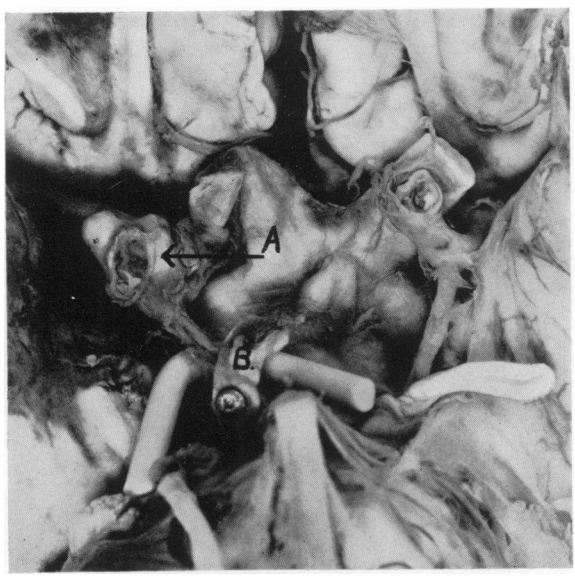

Fig. 2.

Photographs by David Mehaffey.

CASE VII.

A Case of Ruptured Cerebral Aneurysm.

Clinical summary: The patient was a woman of 31 years. She was married and had two children alive and well. Apart from appendicectomy when a child, the previous history is of no importance. Six weeks ago the patient began to suffer from headache. This began quite suddenly, and has always been confined to the right side, though it has varied somewhat in position between the frontal and occipital regions. Since the onset of the headaches there has been frequent nausea, culminating in vomiting when the headache is severe. Vomiting has not been definitely related to food. Ten days ago the right eyelid began to feel heavy, and since then she has been unable to open this eye.

Clinical Examination.

On admission the patient was found to be of average build and fair nutrition. There was no external evidence of anæmia, jaundice, or cyanosis. The pulse at 76 was regular and of good volume. The arterial wall was not palpable. The blood-pressure was 136/92. The heart was of normal size, and the heart-sounds were regular, easily heard, and without adventitious bruits. The lungs were normal. The abdomen, apart from a healed surgical scar over the region of the appendix, showed no abnormality. On examination of the nervous system, the patient was found to be intelligent and perfectly orientated. All the deep reflexes were present. There was ptosis of the right eyelid, but the patient could see perfectly if the eyelid was lifted. The right pupil was fixed and dilated, and there was absence of medial movement of the eye-ball. At one time during the examination the patient complained of diplopia. The left eye was quite normal.

Lumbar puncture showed a blood-stained fluid. The blood was evenly distributed throughout the fluid and did not diminish in intensity as the fluid came away. On laboratory examination, the protein was found to be 0.02 per cent.; globulin, a trace; cells + : blood + ; and the Wassermann reaction negative. The supernatant fluid was colourless.

Following the lumbar puncture, the patient was given an injection of omnopon. She appeared to become comatose, and did not waken until 8 p.m. the following day. She was then very restless and attempted to get out of bed. There was some evidence of seventh-nerve palsy on the right side. She continued in a stuporose state and died the next day.

Post-Mortem.

A limited post-mortem examination was performed. The essential findings were as follows :—

The cranial bones were normal.

On incising the dura mater, blood was seen to extend upwards towards the vertex in the subarachnoid space on the right side. There was extensive hæmorrhage at the base, especially marked over the floor of the third ventricle. Hæmorrhage extended along the sheath of the right third cranial nerve almost as far as its origin.

At the origin of the posterior communicating artery from the right internal carotid was a small aneurysm. This measured 3 mm. in diameter, and from its surface two other small sacculations, each over one millimetre in diameter, protruded. The more lateral of these secondary sacculations was adherent by fine adhesions to the medial aspect of the right temporal lobe. The other more medial sacculation showed a rupture on its inferior surface. Numerous fine fibrous adhesions surrounded the aneurysm, and there was some adherence to the right optic nerve. From the site of rupture the blood had spread laterally along the right Sylvian fissure and backwards into the posterior fossa to cover the right side of the mid-brain and the anterior surface of the right cerebellum. In this manner it had come to bear an intimate relationship to the right seventh and eighth cranial nerves.

On section of the fixed brain, no intra-cerebral hæmorrhage was seen. The Sylvian fissure was greatly widened and distended with recently extravasated blood.

Careful dissection of the other arteries composing the circle of Willis revealed no other lesions. The arteries were everywhere thin-walled and showed no evidence of degeneration.

Anatomical Diagnosis.

Congenital miliary aneurysm of right posterior communicating artery. Rupture : Subarachnoid hæmorrhage : Hæmorrhage into sheath of third cranial nerve with paresis.

Comment.

Aneurysms of the cerebral arteries have been recognised for a long time. In 1761 Morgagni described dilatations of the posterior branches of both carotid arteries. In 1778 Biumi described a ruptured cerebral artery. Gull of Guy's Hospital wrote in 1859 : "Whenever young persons die with symptoms of ingravescent apoplexy, and after death large effusion of blood is found, especially if the effusion be over the surface of the brain in the meshes of the pia mater, the presence of an aneurysm is probable."

A large literature has now accumulated on the subject, and recently MacDonald and Korb have analysed 1,125 cases of saccular aneurism of the cerebral arteries, published after verification at post-mortem or operation. The lesion is by no means uncommon, and in our own material constitutes the cause of death in slightly over one per cent. of all autopsies.

The present case is relatively typical. The patient is usually quite well, when a sudden catastrophe occurs. This may be of varying magnitude. This woman had a sudden onset of a severe headache of very definite localisation, associated with nausea and vomiting which were cerebral in character; that is to say, they were not related to the character or state of the ingested food nor to the time of eating. These are signs of increased cerebral pressure, but the preservation of an active intelligent state shows that this cannot have been of severe degree. The amount of extravasated blood and the reaction to its presence in the subarachnoid space cannot, therefore, have been very great at the onset. Probably only a small leakage has occurred. The appearance of new symptoms ten days before admission, associated with the first appearance of paralysis of the right third cranial nerve, shows that further leakage of blood has occurred, and the post-mortem findings indicate that the nerve paralysis was due to this blood infiltrating the sheath of the affected nerve. The close anatomical relationship between the aneurysm and the third nerve allowed this to happen in spite of the fact that the extravasation of blood was not extensive, for the patient did not lose consciousness, and the increase in the intracranial pressure cannot have been severe. If she had been examined at this time it is probable that some degree of irritation of the meninges by the blood might have been detected. Many of these patients show some stiffness of the neck.

The occurrence of ptosis from this cause is by no means rare. France of Guy's Hospital in 1846 drew attention to this happening, and in several of our own cases in which the aneurysm was situated at the origin of the middle cerebral or posterior communicating arteries, paralysis of one third nerve has been an outstanding clinical finding.

Sooner or later the occurrence of leakage from these aneurysms is followed by a more extensive extravasation of blood leading to a diffuse subarachnoid hæmorrhage and usually death. In the present case, lumbar puncture shows a diffuse staining of the cerebro-spinal fluid by blood. The fact that when the blood was removed by centrifuging the specimen the supernatent fluid was colourless, is of some importance. Often the supernatant fluid shows a slight yellowish discolouration due to the presence of breakdown products of hæmoglobin. The absence of such discolouration in this patient can only mean that the previous leakages were of small volume, and were entirely localised to the vicinity of the aneurysm. The broken-down hæmoglobin would here be phagocytosed by large mononuclears, many of which are trapped in the fibrous adhesions which had formed round the aneurysm. In some patients the organisation of the local extravasation of blood results in the formation of relatively dense fibrous adhesions. These tend to obliterate the subarachnoid space in the neighbourhood of the aneurysm and often bind the affected vessel tightly to the surface of the brain. Subsequently the aneurysm ruptures, and the extravasated blood finds its way more easily into the substance of the brain than through the obliterated subarachnoid space. In this manner quite extensive intra-cerebral hæmorrhages can be produced, even occasionally traversing the brain substance to rupture into the lateral ventricles.

Unless this possibility is borne in mind, the appearance may suggest a primary intra-cerebral hæmorrhage and the aneurysm may be overlooked.

It has been shown by Forbus that these aneurysms occur in vessels which show a congenital deficiency of their media. There is a failure of fusion of the muscle-coat of the branches of the artery with that of the main vessel. Hence these deficiencies are found at the point of junction of vessels. When they occur they are most frequently seen in the angle of bifurcation, and it is in this angle that congenital aneurysms are most common. The site of the aneurysm by no means represents the whole congenital deficiency, for in these cases serial section of the angles of bifurcation of other apparently normal cerebral vessels reveals a similar lack of media. It is not quite clear, therefore, as to what actually produces the aneurysm, but the congenital deficiency offers a reasonable explanation of its site. Similar deficiencies in the media may be found in the branches of the mesenteric vessels, and in one recent case a ruptured cerebral aneurysm was found associated with an aneurysm of the splenic artery and bilateral congenital cystic kidneys.

On the circle of Willis the great majority of the aneurysms are found in an anterior branch. Thus MacDonald and Korb's analysis shows 48 per cent. on the internal carotid or middle cerebral vessels, 15 per cent. on the anterior communicating artery, and only 28 per cent. posterior to the internal carotid arteries.

In spite of the congenital nature of the lesion it is important to appreciate the fact that these aneurysms may produce symptoms at all ages. In our own material the youngest patient was 12 years and the oldest 75 years, but greater extremes of age have been reported. Relatively few aneurysms are found to rupture before the age of 20 years, and it is in the next two decades that the majority cause symptoms.

In a few patients similar small aneurysms are found to be the result of infected emboli, but the histological picture is entirely different.

Other forms of cerebral aneurysm are so rare as scarcely to enter the field of differential diagnosis. Occasionally atheromatous degeneration results in a fusiform dilatation not especially localised to the angle of bifurcation. Occasionally syphilitic change in the arteries may be the cause, but such cases are very rare.

We are indebted to Professor W. W. D. Thomson for the clinical notes.

A Case of Anthrax Meningitis

By H. Hilton Stewart, M.D., M.R.C.P.(Lond.)

Anthrax is a rare disease now, and when a case presents itself accompanied by what must be its rarest complication, it is thought of interest to publish some of the details at length.

A young woman of thirty-two years came up from Dublin with her husband and family to spend a holiday at Bangor, Co. Down. A few days before they were due to go home, the patient complained on returning from the pictures on the 28/6/39 that she was feeling shivery, and on going to bed she took some aspirin.